BRAIN TUMOR SYMPTOM-COMPLEX WITH TERMINATION IN RECOVERY.*

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The title of this paper is not at all misleading to the neurologist. All of us are at times confronted with cases which present the general symptoms of a brain tumor, together with focal signs which seem to render not only the general diagnosis of a cerebral neoplasm probable, but the focal signs are more or less marked, even to such an extent that we have little hesitation in designating the seat of the tumor. After a while, however, perhaps after several remissions and exacerbations extending over a period of years, these cases recover, showing, however, some defect, usually on the part of the eyes. Then death is caused by some other disease, and the brain is found perfectly normal.

Both the cases which recover and those in which a negative condition of the brain is found after death are of sufficient importance to merit our attention.

I will briefly report three cases of this kind, two of which are still alive, and one which died of tuberculosis, and in which an autopsy was made.

Case No. 1.—Cause of death, pulmonary tuberculosis. Under observation 10 years.

Nov. 1, 1895. Alice W., age 23, single, servant. Parents are well, father very nervous. Has sisters and brothers. One sister a confirmed neurasthenic with obsessions. No history of syphilis nor any evidence whatever of it to be found on examination. Patient has been well with the exception of an attack of la grippe two years ago. Has had attacks of headache for 4 years with occasional nausea. These attacks came on at night especially. Two years ago headaches became very severe, had enlarged glands in the neck at this time which were very painful. At this time the pain was constant, located in the occipital region, associated with pain in and behind both eye-balls. Has attacks of jerking in the neck and back.

The headaches are subject to acute exacerbations, during which patient walks the floor, tears her hair, screams and "seems to lose her mind." During this time patient's character changed. She became cross, querulous, was often unmanageable, subject to fits of violent temper. There was some weakness of both legs and staggering gait. The vision was diminished. These violent headaches and other symptoms would last two or three months. Then there would be a period of freedom from pain followed by another siege of headache, etc. Seven months ago she began to lose sight in the left eye. Could not distinguish objects. Legs were often numb and cold. During entire summer the legs were weak. Staggering gait came on at this time and has continued ever since, being worse on arising in the morning and when fatigued.

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Examination.—Patient is of medium height, well built, 120 lbs. in weight. Intelligence good, memory good. Has violent headaches. Vertigo when she leans forward. Typical choked disk. Pupils equal and react to light, marked choked disk. No derangement of external muscles of eyes. Vision of left eye almost gone, has only light perception. Slight weakness of right facial. Has typical staggering gait (cerebellar). Muscular power and sensation normal. Left patellar reflex absent, right present.

Diagnosis.—Cerebellar Tumor(?).

Patient was treated with iodide of patassium and later on with proto-iodide of mercury.

The symptoms gradually disappeared after six months. Has headache about once a week. Choked disks had disappeared, was able to read large print with left eye, good sized scotoma in upper and outer part of field. General weakness of arms and legs but can walk well, no staggering gait.

Left patellar reflex absent, right normal.

A few months later had slight horizontal nystagmus, knee reflex returning on left side. Complains of buzzing in the head.

After a total illness of three years, and about 14 months after beginning of treatment, patient returned to her occupation, being entirely well with the exception of some defect of vision in the left eye.

Patient remained entirely well for six years.

July 9, 1902, aet 30. Has been at work 7 years. Patient returned with statement that she occasionally had headache, insomnia, vertigo, but no vomiting, these symptoms would last but a short time and would disappear.

Lately the headaches had again become more violent, they were frontal, lateral and occipital, with occasional vomiting. Staggering gait, states that she cannot see with left eye at all and only very little with the right. General weakness, can come to office.

Examination.—Some mental hebetude, peculiar inclination of head to right side probably due to defect of vision.

Marked choked disks of both eyes. The examination is otherwise negative. Patient was again placed on iodide of potassium.

In six or eight weeks all the above signs and symptoms disappeared again. About one year later patient developed an ordinary case of pulmonary tuberculosis.

I saw her once during the three years preceding her death. She became mentally unbalanced. Mental confusion, became violent at times because of suspicions of food being poisoned. This period lasted for several months and gradually passed away. The examination carefully made showed that there was an absence of all signs and symptoms pointing to any organic brain lesion.

Died Dec. 20. 1905. The autopsy was limited to an examination of the brain. The brain was perfectly normal, normal quantity of cerebro-spinal fluid. Dura mater and pia mater normal. Nowhere any adhesions. Cortex carefully examined microscopically was normal in every convolution. The ventricles were absolutely *not* dilated, the basal ganglia normal. Cerebellum was most carefully examined, no cicatrices, no atrophy, no adhesions on surface of the cerebellum, fourth ventricle, peduncles, pons, and medulla are all normal. The canal of Sylvius was open. The foramen of Magendic was also normal.

Hence no signs of a meningitis, hydrocephalus or brain tumor or abscess anywhere to be found.

CASE II. Under observation 13½ years.

Kate E., aet. 16, Buffalo, W. Va., was first examined by me on Dec. 30, 1892.

Family History.—Father alive and well. Mother dead, cause pulmonary tuberculosis. Has two sisters and one brother, all of whom are well. Collateral family history negative.

Present trouble began in 1888, four years ago. Previous had typical attacks of migraine or which were supposed to be such, viz., headache, vomiting and general prostration, the attack lasting usually 24 hours. Since onset of present trouble she has not had any sick headaches.

Present trouble began with difficulty in hearing and seeing. The difficulty in hearing manifested itself in gradually increasing deafness, which began four year ago. At the same time there was failing eye-sight in the right eye.

Menstruation, previously regular, has ceased for past ten months.

Vision has gradually diminished in right eye until patient is unable to see at all for past three years.

Turning of head to left causes pain in head and below the ears, complete turning of the eyes upward also causes pain in head.

Has had vertigo and vomiting for the past three years. Somnolence is very marked, is sleepy, sleeps often during the day.

Sleeps soundly at night. Never loss of consciousness, frequent attacks of staggering with the vertigo. Has been deaf in right ear for past three years:

About one year ago noticed that she could not turn right eye outward, lately has had daily headaches.

Physical examination.—Mental condition good, facial expression good.

Vision, right eye 0.7; left eye 1.0. Pupils equal in size and react to light.

Bilateral optic neuritis, choked disk, unable to close right eye completely. Paresis marked of right abducens, slight weakness of left abducens.

Head turns to left, face is inclined to left. Entire body inclines to left side. No tenderness of calvarium on percussion. Paresis of right side of face. Tongue is protruded in median line.

No disturbance of sensation anywhere.

Arms normal.

Legs, muscular power and reflexes are normal.

Marked static ataxia, cannot stand on right foot with closed eyes, very difficult to stand on left foot. Staggers to left when walking.

Dr. Ayres' examination.—Right eye, V = 0.7, left eye, V = 1.0.

Right ear loud tones 2 inches. Left ear hearing normal. Loud tinnitus.

Right optic disk presents a rounded mass which extends over and obscures the edge of disc. The veins are large, arteries obscured by the swelling.

Left optic disk is enormously swollen, no hemorrhage into the retina.

Patient was placed under treatment of iodide of potassium. Returned home in January, 1893, and remained entirely well for a year (see letter). She had an attack of fever in February, 1894. In May, 1894, again became very ill with a headache and returned to Cincinnati in June, 1894.

Examination, June, 1894.

Paralysis of external rectus of right eye. Vision, right eye 0.2, left eye 1.0. Marked choked disk both eyes.

Great difficulty in walking, cannot walk unassisted, staggers and falls to the left.

Has severe pain in the left side of head and discharge from the left ear, no evidences of mastoid disease.

Has severe pain in left occipital region.

In a letter dated March 9, 1906, patient writes that she has recovered entirely. She is strong and well and capable of personally doing the housework for her father. There remained only some defect of vision of the right eye and a partial paralysis of the right external rectus. For the relief of the latter she had performed a tenotomy in April, 1905, which was completely successful, relieving the strabismus which had been present since 1894.

Viewing this case critically, in 1894 we have a history of some cerebral process gradual in its onset, gradually increasing in intensity, gradually compressing the cranial contents producing headache, vertigo, vomiting, somnolence, choked disks as general manifestations of a brain tumor and amblyopia of right eye, paresis of right external rectus, paresis of left facial, typical staggering gait, falling to left and static ataxia especially when standing on left foot.

A diagnosis of cerebellar tumor was certainly justified considering the slow gradual onset in a healthy robust girl, together with the absence of any signs of tuberculosis anywhere, or any focus of pus which might have produced a cerebellar abscess. (The otorrhea did not develop until 5 years after onset of first cerebral symptoms.)

Case III. Under observation 2 years, sent by Dr. B., Georgetown, Ky. Examination August 22, 1904.

J. W. B., male, farmer, aet. 47, American, married, 4 children, all healthy. Patient denies all venereal diseases. Has always been well and worked hard. During past four or five years has had stomach ache with frequent attacks of vomiting. Has not been a drinker. Has had violent headaches for past four months, occipital in character. He had two attacks of violent vertigo in both of which he fell to the ground with momentary loss of consciousness. Has double vision very often. Cannot recognize objects one hundred feet off. Is unable to read ordinary print. Vomits occasionally. Has occasional attacks of staggering, as if he were drunk.

Examination.—Facial expression heavy, mental hebetude well marked. Intellect good. Memory good.

Pupils equal in size and respond to light. Marked choked disk in both eyes. External muscles of eyes normal. Reflexes all normal. Examination in every other respect negative.

Résumé: Headache, four months in duration, vomiting, violent vertigo with attacks of momentary unconsciousness. Staggering gait, double vision, great diminution of vision and bilateral choked disk.

Diagnosis.—Probable cerebellar tumor.

Examination May 1, 1906 (has not been examined for twenty months). Patient has gradually improved especially so in the past eight weeks. But made no improvement at all until six months ago. Headache has disappeared.

Has still occasional attacks of vertigo, slight staggering but very mild compared to formerly. Double vision is still present, objects being side by side. Can read large print.

Examination.—Mental condition bright, considers himself practically well. Has done some work this spring. Pupils equal in size and react to light.

Slight atrophy of left optic nerve. Right seems normal.

Right patellar reflex diminished, left normal.

Examination in other respects negative.

This patient has been taking iodine in some form all this time. There seems but little doubt that this case belongs in the same category as the other two.

The diagnosis of brain tumor is based upon the presence of certain symptoms and signs which point to a gradually increasing local and general compression of the cranial contents.

Of these the general signs and symptoms together with choked disk are of greatest importance in making the diagnosis of the presence of a tumor. The chief factors in estimating the value of these signs and symptoms are:

First—That of Time. Second—Compression.

As to the factor of time we must determine that from slight and insignificant beginning the symptoms gradually in the course of months grew in severity, duration and number, perhaps subject to periods of remissions and exacerbations, but nevertheless steadily growing worse as time passed by. The second point is equally important. All the symptoms must be the result of a gradually increasing compression of the cranial contents with localized destruction of brain tissue. When those general and local symptoms occur in sufficient number we can be reasonably certain of the presence of a tumor, and ordinarily the diagnosis and localization are not difficult. The only outcome of the brain tumor is relief by surgery or death, and then the diagnosis is usually verified. Leaving out of consideration possible errors of diagnosis which are shown up by autopsy, when abscess, hydrocephalus or some other gross lesion of the brain has simulated a brain tumor; there remain a few cases, as undoubtedly has happened in the practice of us all, in which, after careful examination, when all the signs and symptoms pointed to the presence of a brain tumor, the patients either recovered with perhaps only a slight defect or, death being caused by some other intercurrent disease. the autopsy revealed a negative condition of the brain.

Cases of this character are interesting enough, and of sufficient importance to demand our attention. While every one of us has undoubtedly seen them, the literature is exceedingly scanty. There are but two papers which deal extensively with this subject. I refer to the articles of Nonne and Oppenheim. Nonne's article gives in detail 8 cases occuring in adults and Oppenheim's deals with a similar condition in children.

It is needless to say that cases such as these cannot be attributed to a functional derangement of the nervous system. I may say that we can rule out hysteria at once. Such an array of general and local symptoms can be due only to some organic change either in the brain itself or in its membranes, and this lesion must be capable of complete retrograde change, restoring the brain to its original normal condition.

In considering the possible pathological condition underlying these cases we must divide them into two groups:

(a) Those cases in which death has occurred from some accidental cause and in which the autopsy revealed no brain lesion whatsoever.

(b) Those cases in which recovery occurred and the patients are still alive or having died, no autopsy was held.

There are very few cases of the first group on record. Nonne publishes three cases of this kind with autopsy in which the brain examination, including microscopic and bacteriological examination was entirely negative. Since Nonne's publication (1890) I have found no others and I wish to put my case with negative autopsy on record. While Nonne declines to consider the possibility of hydrocephalus acquisitus or serous meningitis, but inclines to the theory that some unknown and hitherto undiscovered organic lesion which is perfectly curable must be looked upon as the cause of the trouble, it seems to me that we must consider one of the following conditions as the underlying cause, I refer:

1. To serous meningitis producing acquired internal hydrocephalus, and
2. Hemorrhagic non-purulent encephalitis with serous effusion.
3. Chlorosis.
4. Chronic cerebritis.

In the purely clinical cases, which have recovered after all the signs and symptoms of brain tumor were present, we must consider in addition to the above condition, the possibility of

5. Brain tumors or tubercles which have either undergone a retrograde metamorphosis or have become arrested in their development and to which the brain has accommodated itself.

It has been shown that all of the above pathological conditions except cerebritis, may produce a symptom-complex not unlike that of brain tumor and terminate in recovery.

Let us take up internal hydrocephalus first. There can be no doubt to-day, first, that internal hydrocephalus or serous meningitis occurs quite frequently in the adult, and in the second place it can hardly be doubted that although most of these cases terminate fatally, recovery may and does take place. Quite a number of acute mastoid inflammations are associated with a meningitis which disappears in a day or two after the operation, and which is in all probability serous in character.

It is possible that the majority of cases with brain tumor-symptom complex which terminate in recovery are really cases of serous meningitis of a subacute or chronic character, leading to internal hydrocephalus. We know now, that acquired hydrocephalus not only produces the general signs of a brain tumor, such as headache, vomiting, vertigo and choked disk, but also focal signs, such as staggering gait, hemiplegia, aphasia and paralysis of the various cranial nerves.

Oppenheim calls attention to the fact, that it is difficult, and often impossible, to differentiate between a tumor of the occipital fossa and meningitis serosa, and that the latter condition is the most frequent cause of a mistaken diagnosis of a cerebellar tumor. He says further, that these cases usually occur in women who present the following symptom-complex: Headache, vertigo, vomiting, choked disk, paresis of the external recti muscles, more especially of the right eye, nystagmus, more especially when the eyes are turned outward, especially toward the paretic muscle, areflexie or hyporeflexie of the cornea, tinnitus and diminished hearing on the same side, and cerebellar ataxia. He looks upon this symptom-complex as typical, and says that whereas in the beginning he always made the diagnosis of cerebellar tumor on the affected side, he now knows that these are cases of acquired internal hydrocephalus or serous meningitis. The apparently focal signs of internal hydrocephalus are partly due to distal pressure, and this would explain the paralytic signs on part of the cranial nerves at the base of the brain. Oppenheim says that a unilateral predominance of pressure in the labyrinth of one side may so affect the cochlearis and the nervus vestibularis as to cause staggering to one side, tinnitus, as well as the nystagmus, and the abnormal holding of the head.

As a rule, gross unilateral symptoms on part of the extremities are absent in acquired hydrocephalus, but they may be present, as we know from reported cases, prominent among which is the case of unilateral hydrocephalus reported by Spiller. These, however, are the exceptions, and when they do occur they are not progressive nor permanent (Oppenheim). The focal signs may also be explained by the fact that serous meningitis may be complicated by a mild localized encephalitis or, what may also happen, the encephalitis may be the cause of the serous meningitis. Monoparesis, hemiparesis and hemianopsia are rare, still they have been observed (Annuske, Quinke and Kupferberg).

Moreover, we may have attacks of convulsions with periods of somnolence and coma, lasting perhaps a few days and then disappearing, perhaps not to return in months. I have seen this in a case of hydrocephalus which was associated with a brain tumor, and in which these attacks occurred six or eight times in a period of two or three years. Remissions are more common in hydrocephalus than in brain tumor. Herzfeld calls attention to these attacks coming on with almost apoplectic suddenness after long periods of comparative well being. Oppenheim attributes these attacks to a sudden increase of intraventricular pressure, or to a sudden compression of the medulla or vasomotor centers caused by a change in the position of the head.

We see, therefore, that there is a marked similarity not only between acquired hydrocephalus and cerebellar tumors, but also tumors of the brain in general. As a rule time will clear up the diagnosis, and even during life we can say with considerable certainty whether we have tumor or hydrocephalus. If the patient dies, then as a rule, the condition becomes certain. But what of those cases in which the autopsy is entirely negative or in which the patients recover and remain well? Nonne concludes that in the eight cases of brain tumor complex with recovery which he reports in his article, there could not have been an acquired internal hydrocephalus or miningitis serosa, because of the absence of the etiological factors of the latter condition, namely: alcoholism, physical or psychical shock, insolation and infection.

It is possible, however, to have acquired hydrocephalus without any cause known or discoverable during life, and I personally am inclined to think that at least some of these cases are due to acquired hydrocephalus taking its origin from some cause unknown during life, which is either favorably affected by the treatment or disappears spontaneously, the exudation being resorbed.

Let us consider briefly the etiology of acquired internal hydrocephalus in regard to its pathology.

Spiller reports a case occurring in the service of Dr. Mills in which during life the diagnosis of cerebellar tumor was made. The autopsy however revealed an internal hydrocephalus caused by closure of the aqueduct of Sylvius by proliferation of the neuroglia. Byrom Bramwell reports a similar case caused by a closure of the foramen of Magendie. J. Parkes Weber, reports a case of acquired hydrocephalus with marked ependymitis of the fourth ventricles. He draws an analogy between acquired hydrocephalus and serous pleurisy and peritonitis. In both of these conditions we may have a large serous effusion caused by a small local area of inflammation, and he asks whether a large acquired hydrocephalus may not be caused in the same way. This conclusion would seem to be borne out by an autopsy which I recently made on a young boy, in which a marked hydrocephalus complicated a case of brain tumor. The tumor, an endothelioma, grew in the thalamus and extended into the white matter. Covering the serous surface of the lateral ventricle just over the tumor was a small area of tough fibrous exudate about the size of a silver half dollar which could be peeled off. In this case the symptoms were typical for acquired internal hydrocephalus, and antedated the focal signs of the brain tumor for years. It is not improbable to conclude that this hydrocephalus may have had its origin from this small area of inflammation which was found located over the tumor in the lateral ventricle.

We also know that autopsies do not always show a closure of the various foramina in internal hydrocephalus.

From the reports of well examined cases we know that acquired hydrocephalus may occur from an inflammation of the tela choroidea, the choroid plexus, from pressure upon the vena Galeni, or a localized inflammation around the opening of the fourth ventricle. Any of these conditions might occur during life, without our knowledge, and terminate in recovery. Anton says that a frequent cause of chronic acquired internal hydrocephalus is a localized meningitis at the base of the brain. Quinke and Bonninghaus say that this meningitis spreads to the tela choroidea and the choroid plexus, and in this way causes internal hydrocephalus. Any localized swelling or inflammation around the foramen of Magendie or along the course of the canal of Sylvius may lead to a closure of these passages and thus cause an internal hydrocephalus. These various localized inflammations may yield to treatment, or heal spontaneously. The passages may reopen and the accumulated fluid become resorbed and the brain may return to a normal condition.

I refer to Anton, Quinke, Gowers and Oppenheim as authorities, that we may have recovery in typical cases of internal hydrocephalus acquisitus. The symptom-complex which is at times seen in myxedema of adults is probably due to a temporary meningitis serosa or internal hydrocephalus. Thus, I have a patient who has had myxedema for many years. She is very intelligent and highly educated. For years she has had attacks which begin with violent headache, vertigo, irritability, mental and emotional depression, marked staggering gait, delirium, mental confusion and, finally after some days, inability to walk, terminating in great somnolence, apathy and apparent dementia. These attacks may last from three to five days, to as many weeks, and then terminate rather quickly when the thyroid extract is pushed. She has had from one to four attacks per year for the past five or six years, and I look upon them as being due to an internal hydrocephalus, caused perhaps by the same toxin which causes the general myxedema. I therefore feel that a large percentage of the cases of so-called brain tumor ending in recovery are really subacute cases of internal hydrocephalus which terminate in resolution, I include among these, those cases in which death has occurred and in which the autopsy has been negative.

Have we any means of making the positive differential diagnosis between brain tumor and acquired hydrocephalus in these doubtful cases. It is needless to say to-day that the Quinke puncture, or the specific gravity of the fluid will not clear up the differential diagnosis. The factor of time itself will only enable us to say in the recovered cases, or in cases with a negative autopsy, that we did not have a tumor but may have had an internal hydrocephalus.

Polio-encephalitis hæmorrhagica usually is acute in its onset and rapid in its course and does not ordinarily cause a symptom-complex resembling brain tumor. But the reported case of Schultze shows us how a polio-encephalitis superior may simulate a tumor of the corpora quadrigemina especially if it is complicated with an ependymitis with a secondary internal hydrocephalus. Schultze's case had in addition to the focal symptoms, somnolence and optic neuritis. Oppenheim reports a similar case with recovery, and he states that he had had a number of such cases, one in which he had even advised an operation, and recovery occurred. He concludes that there was present either a nonpurulent hemorrhagic encephalitis or some hitherto unknown pathological process capable of complete recovery.

Cases of completely cured polio-encephalitis, with perhaps some slight defect, do undoubtedly occur and we have thus another condition which may cause all the signs and symptoms of a brain tumor and terminate in recovery. In these cases, according to Oppenheim, the localized encephalitis is in or near the cortex, which explains the occurrence of Jacksonian epilepsy, monoplegia, aphasia, etc.

In this connection I also wish to refer to meningitis tuberculeuse en plaque which Oppenheim looks upon as the cause of brain tumor symptom-complex occurring in a number of children who recovered, and the author looks upon the localized tubercular meningitis as a curable affection.

Therefore, as far as encephalo-meningitis is concerned, we can never be absolutely sure of the diagnosis, because we cannot rule out with certainty either localized tubercular or localized syphilitic meningitis.

We know from the recorded cases of Crawford-Thompson, Burton-Fanny, Jollye, Gowers, that chlorosis may produce a symptom-complex which may temporarily simulate brain tumor. We have in these cases pronounced headache, vomiting, vertigo, together with optic neuritis. Oppenheim reports a case of especial interest, in which the anemia was a complication of carcinoma of the breast, and the headache, vomiting, vertigo and optic neuritis simulated a metastatic corcinoma of the brain. The importance of recognizing the fact that anemia may produce a brain tumor symptom-complex should render us more careful about vetoing the amputation of a breast which otherwise might not only prolong the life of the patient, but even end in recovery.

The consideration of pseudo-tumors of the brain, with negative autopsy cannot be complete without taking into consideration the subject of chronic cerebritis or cerebral hypertrophy. This condition is exceedingly rare, and occurs most frequently in children, but may occur in adults. In adults where the cranium cannot expand we may have all the signs of intracranial pressure with irritation, resembling those of brain tumor. The cases are acute in their course, but may be subacute. The symptoms, according to Eulenberg, resemble very much those of acute hydrocephalus, violent headache, vomiting, slowness of the pulse, general convulsions, perhaps optic neuritis.

Let me give you Rokitansky's description of this condition: If we split open the dura mater we perceive immediately that there is a swelling of the brain, so much so, that there is great difficulty in putting back the calvarium. The various membranes are remarkably thin, the dura mater is tender, pale red in appearance, the pia mater is very close on the one side to the dura, on the other to the cortex. They are abnormally dry and their vessels small, flat and unusually free from blood, the brain hemispheres appear at first sight unusually large, the convolutions are pressed close together and flattened to such an extent that the sulci are hard to recognize. The white substance of the brain which has increased in volume is pale, anemic, which differentiates this condition from hyperemic turgescence which Rokitansky looks upon as due to an increase in the neuroglia. Anton holds that we may have also a parenchymatous hypertrophy of the brain. Rokitansky and Eulenberg look upon the condition as due to a general increase of the neuroglia.

Eulenberg says that the fatal termination is usually due to a congestive hyperemia.

The literature on this subject is exceedingly scanty, but if such a case should occur and we were on the lookout for a tumor, especially if the brain were presented to us after the removal from the calvarium, the condition might easily be overlooked and the autopsy considered negative.

Anton asserts that there may be a partial cerebral hypertrophy, which if it did occur might produce even a more decided brain-tumor symptom-complex. He gives, however, no data to substantiate this assertion.

In the cases of recovery from brain-tumor we must bear in mind that the patient may really have had a brain-tumor, and that the latter disappeared, or the brain became accustomed to its presence after it ceased to grow. Some of these cases, however, are cases of brain-tumor with unusually long remissions of the symptoms and not cases of real recovery, which like Osler's case finally end fatally. Oppenheim, Gowers, Bernhardt and Rüssel have reported similar cases. Psammomata, lipomata, cholesteatomata may attain a certain size and then cease to grow. Cysticerci and echinococci may die and shrink. The observations confirmed by autopsy are exceedingly rare. Nonne was able to find but four cases, those of Bruns, Oppenheim and Simeon.

Aneurysms may cease to grow or become obliterated by the formation of a clot and the brain accommodates itself to the pressure (Oppenheim, Hutchinson, Hodgson and Humble). Solitary tubercles may undergo caseation calcification and then become encapsulated causing no further damage (Wernicke, Starr, Knapp, Gowers, Babinski and Sahlberg). These observations are confirmed by the pathological observations of Simeon who reports an autopsy in which a calcified tubercle was found which must have been in the brain for 30 years. Kirschberger's case shows how a tubercle may show all the signs of a brain-tumor for a year and a half and the patient recover and remain well for six and one-half years. The autopsy in this case showed that the tubercle had become calcified. Gummata may become absorbed and disappear.

Long remissions and even permanent disappearance of symptoms may occur in brain-tumor as numerous cases on record show. These cases, however, would hardly come under the head of pseudo-tumors with a complete disappearance of symptoms, because they had been regarded during life as cases of epilepsy of many years' duration, and after death partly or completely ossified tumors had been found as the cause of the epilepsy.

That a pathological mass may be present in the cortex of the brain for 40 years and cause no interference with the ordinary daily life of an individual the following case will show:

Lately an individual, 60 years of age, died in my service at the Cincinnati Hospital from acute meningitis. He states that at the age of 21 his left arm became paralyzed, but that it soon recovered. He has lived the life of a farmer and teamster for the past 40 years. With the exception of some weakness of the left arm and a constant headache he had always been well.

The autopsy revealed a mass, hard, indurated, cartilagenous, about the size and shape of a peanut, situated in the cortex, at the posterior end of the first frontal convolution of the right side, pressing upon the ascending frontal convolution. This mass was partly cortical and partly subcortical, was not firmly adherent, and was surrounded by recent softening. On splitting it open we find a very hard stony calcareous center and a hard cartilagenous covering.

The practical conclusion of the consideration of this subject is, in the first place we should not be too pessimistic in our prognosis of brain-tumors, especially in cerebellar cases, until a considerable period of time has elapsed; and in the second place we should not be too hasty in the recommendation of surgical interference both in children and in adults, until therapeutic measures have been given a long and complete test.

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